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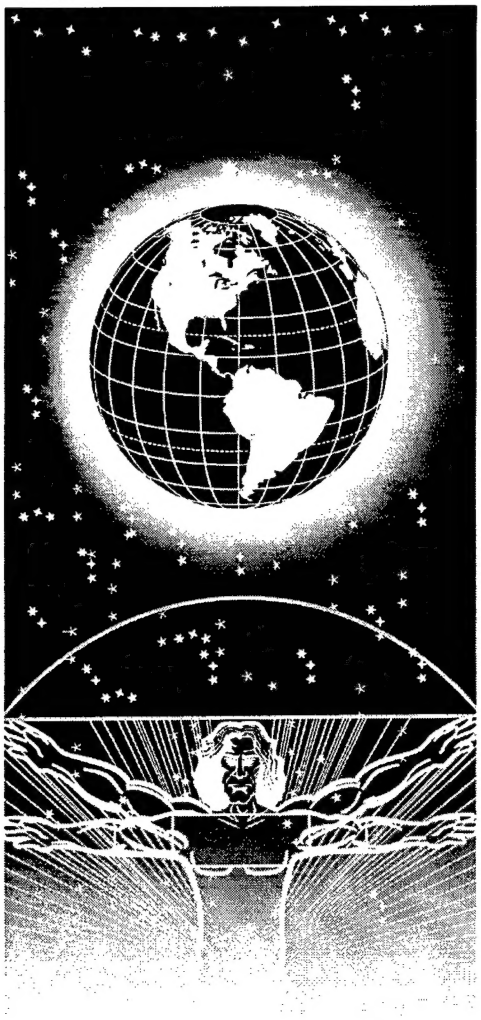

UNITED STATES AIR FORCE RESEARCH LABORATORY

EFFECT OF G-SUIT PROTECTION ON CORATID-CARDIAC BAROREFLEX FUNCTION

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This technical report has been reviewed and is approved for publication.

Victor A. Convertino

VICTOR A. CONVERTINO, Ph.D.
Project Scientist

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To test the hypothesis that G-suit inflation could increase cardiac chronotropic responses to baroreceptor stimulation and enhance baroreflex buffering of blood pressure, the carotid-cardiac baroreflex response of 12 subjects was measured across two levels of lower body negative pressure (LBNP = 0 and 50 mmHg) and two levels of G-suit inflation (0 and 50 mmHg) in random order. Carotid-cardiac baroreflex stimulation was delivered via a silastic neck pressure cuff and responsiveness quantified by determination of the maximum slope of the stimulus-response function between R-R intervals (ms) and their respective carotid distending pressures (mmHg). Mean ± SE baseline control baroreflex responsiveness was 3.8 ± 0.4 ms/mmHg. LBNP reduced the baroreflex response to 2.7 ± 0.4 ms/mmHg ($P < 0.05$), but G-suit inflation, both with and without LBNP, restored the baroreflex response to 4.4 ± 0.7 and 4.3 ± 0.6 ms/mmHg, respectively. These results suggest that, in addition to increased venous return and elevated peripheral resistance, G-suit inflation may provide protection against the debilitating effects of blood distribution to the lower extremities during G acceleration by increasing cardiovascular responsiveness to carotid baroreceptor stimulation.

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INTRODUCTION

Failure to maintain adequate cerebral perfusion during maneuvering in high-gravity (G) military aircraft could lead to catastrophic incapacitation of crew members. The technological development of special pressure ensembles (G-suits) designed to counter the effects of head-to-foot G forces (+Gz) during high acceleration maneuvers has provided significant protection against G-induced loss of consciousness (G-LOC). The hypothesis has been advanced that a primary mechanism of G-suit effectiveness represents a mechanical hydrolic effect of defending blood pressure through increased vascular resistance and enhanced venous return, with little role of baroreflexes due to their relatively slow response during unprotected rapid onset rate (ROR) of +Gz (1,2). In contrast to this notion, invasive measurements of beat-to-beat hemodynamic responses clearly indicated that recovery of arterial blood pressure occurred within the 4 to 6 seconds following ROR acceleration despite diminished cardiac output, suggesting the contribution of baroreflex-mediated compensation (18).

Recent data indicate that heart rate response to carotid baroreceptor stimulation is attenuated by head-to-foot fluid shifts induced by standing (15,17). In contrast, heart rate response to carotid baroreceptor stimulation is accentuated when foot-to-head fluid shifts are induced by pressure applied to the lower body (9). Taken together, these observations might reflect that a functional role of G-suit protection is to provide a capability to maintain an optimal physiological range of baroreflex response by minimizing or eliminating the footward fluid shifts that reduce carotid-cardiac baroreflex responsiveness. The protection of vagally-mediated carotid baroreceptor reflex control of heart rate may prove important to maintenance of arterial pressure and cerebral blood flow during high +Gz exposure since this baroreflex has a rapid response time (6) and has been shown to be associated with orthostatic performance (4,12) including at high +Gz acceleration (14). The purpose of this study was to investigate the direct interaction between head-to-foot and foot-to-head fluid shifts and the normal carotid-cardiac baroreflex response in humans. We hypothesized that the heart rate response to carotid baroreceptor stimulation would be attenuated (i.e., baroreflex responsiveness would be reduced) during head-to-foot fluid shifts and would be reversed with counter foot-to-head fluid shifts induced by G-suit inflation.

METHODS

Subjects. Twelve healthy, normotensive, non-smoking men with a mean ± standard error (SE) age of 33 ± 2 years, weight of 77.73 ± 2.6 kg and height of 176 ± 5 cm served as subjects. The subjects were not pilots nor had they undergone any particular type of exercise training. Individuals taking prescription drugs were excluded from the study. Because of the potential effects on baroreflex function, subjects refrained from exercise and stimulants such as caffeine and other non-prescription drugs 48 hours prior to testing. During an orientation period that preceded each experiment, all subjects were made familiar with the laboratory, the protocol, and procedures. Each subject gave their written voluntary consent to participate. Experimental procedures and protocols were approved by the Advisory Committee on Human Experimentation at Brooks Air Force Base.

Protocol. We used a 2x2 factorial experimental design conducted in 12 randomized blocks (subjects). The vagally-mediated cardiac response (i.e., R-R interval) to specific carotid baroreceptor stimulation was measured in our 12 subjects with and without application of lower body negative pressure (LBNP) and G-suit inflation to determine if fluid redistribution from the lower extremities toward the heart associated with G-suit inflation would reverse the inhibition of baroreflex control of heart rate induced by fluid accumulation in the lower extremities caused by +Gz. Baroreflex sensitivity was measured under four experimental conditions: 1) 50 mmHg of LBNP; 2) 50 mmHg of LBNP with 50 mmHg of G-suit inflation; 3) 50 mmHg of G-suit

inflation without LBNP; and 4) No LBNP or G-suit inflation (control). Consequently, we had two factors (LBNP and G-suit inflation) each at two levels (present and absent). Each subject received all four treatment conditions during a single experimental session, with the order of treatment systematically counterbalanced with three 4x4 Latin squares (3). Application of LBNP and/or G-suit inflation was continuous during each treatment, but was discontinued between treatments. The total time of each treatment was 5 min to allow for the collection of baroreflex data and there was 5 min of no purturbation of the subject in the supine posture between treatments. Stimuli to carotid baroreceptors (neck chamber pressure) were accurately controlled and reproducible (Table 1). Testing of baroreflex responses was initiated after one minute of LBNP and/or G-suit inflation to begin measurements only after adequate time was provided for reflex adjustment of blood pressure. Our experiment therefore allowed us to examine two main effects (fluid distribution toward the legs (LBNP) and fluid distribution toward the head (G-suit inflation)) and their interaction. A statistical power analysis was performed to determine optimum sample size when encountering a medium size effect. Variance estimates from previous studies involving the measurement of carotid-cardiac baroreflex sensitivity were used to determine sample size for a minimum statistical power of 0.80 (5,13). All power calculations were performed with the NCSS Power Analysis and Sample Size computer program (Kaysville, Utah: Jerry Hintze, 1991).

G-suit and LBNP instrumentation. Figure 1 presents a line drawing of the experimental apparatus set-up. Each subject was fitted with the standard Air Force anti-G suit (USAF CSU-13B/P). The anti-G suit consisted of five interconnected bladders (2 thigh, 2 calf, and 1 abdominal) with inflow at the abdominal bladder. The anti-G pants were fitted and secured around the legs and abdomen so they applied equal pressure throughout the lower extremities during inflation without constricting during non-inflated data measurement periods. Subjects were then instrumented with electrode tape (Label Technologies, Inc., Atlanta GA) around the neck and abdomen just below the xyphoid process. Each subject was then placed in the supine posture with both legs inside an LBNP chamber and an airtight seal formed at the iliac crest by a specially-designed neoprene skirt (M.E.S.A., Inc., San Antonio TX). An automated brachial blood pressure cuff (Colin Medical Instruments, Corp., Komaki, Japan) was placed on the right arm for measurement of blood pressure and a lead-II electrocardiogram (Gould, Inc., Cleveland OH) provided continuous measurement of beat-to-beat R-R intervals. Once the LBNP chamber was sealed and instrumentation was verified, a Barocuff neck chamber (Engineering Development Laboratory, Inc., Newport News, VA) was placed on the neck and attached to a computer-driven bellows system (Engineering Development Laboratory, Inc., Newport News, VA) used to apply positive and negative pressure to the carotid arteries. This system, which included data acquisition, was used to measure baroreflex responsiveness.

Carotid-Cardiac Baroreflex. During each of the experimental conditions, carotid baroreceptor-cardiac reflex responses were measured by applying stepwise pressure changes directly to the carotid baroreceptors with a special neck cuff and simultaneously measuring the reflex heart rate response (5,13,19). A stepping-motor driven bellows was used to deliver a series of pressure and suction steps to a silastic neck chamber. At mid-expiration, the subject held his breath and a pressure of 40 mmHg was delivered to the chamber and held for 5 R-waves; then, with each successive R-wave, the pressure was sequentially stepped to +25, +10, -5, -20, -35, -50, and -65 mmHg, before being returned to ambient pressure. Pressure steps were triggered by R-waves so that neck-chamber pressure changes were superimposed upon naturally-occurring carotid pulses. This timing was chosen so that experimental baroreceptor stimuli were as physiologic as possible. With this technique, arterial pressure changes are small during neck chamber pressure changes (19). Systolic blood pressure was measured by auscultation during the last minute of each of the four treatments and carotid pressure was estimated as systolic pressure minus neck-chamber pressure applied during the heart beat. During each of the four experimental conditions, the stimulus sequence was repeated 5 times and the data averaged for each subject. Previous studies in our laboratory indicate that baroreceptor stimulus-sinus node

response relations, measured in this way, are highly reproducible (13). R-R intervals for each pressure step were plotted against estimated carotid distending pressures. The resultant plot was used to define responsiveness (maximum slope determined by least squares linear regression), carotid pressure at maximum slope (point of maximal buffering halfway between the pressures which define the maximum slope), and the operational point (the percent of the total response function that falls below baseline R-R interval).

Stroke Volume. The four silver tape electrodes, two placed around the neck and two placed around the thorax, were wired to a Minnesota Impedance Cardiograph (Model 304B, Surcom, Inc., Minneapolis MN) for non-invasive rheographic determination of beat-to-beat stroke volume (11). This instrument introduced a frequency of 50 kHz at a low constant current (1 mA R.M.S.) into the thorax through the outside electrodes and detected changes in electrical impedance of the thorax with each pulse beat across the inner pairs of receiving electrodes. Five impedance waveforms were recorded during the initial 10 sec of each minute of each treatment condition and the resulting twenty-five waveforms were used to determine the average stroke volume for each 5-min treatment period. Stroke volume was calculated from each waveform as the product of the resistivity of blood (calculated from hematocrit), the average distance between the inside electrode bands, the baseline impedance of the thorax, the ejection time (measured horizontally on the impedance cardiogram from the start of steep upstroke to downward deflection at the end of ejection), and the amplitude from the baseline to peak of the impedance cardiogram tracing. Cardiac output was calculated as the product of heart rate and stroke volume and systemic peripheral resistance was calculated by dividing mean arterial pressure by cardiac output.

Statistical Analysis. Standard descriptive statistics were performed on each of the response variables of interest, with results presented as means ± SE. The effects of LBNP and G-suit inflation, each at two levels (absent and present), were evaluated with a 2 x 2 randomized block analysis of variance for each dependent effect. The experimental design permitted the removal of subject-to-subject variation (subjects acted as blocks) since each of the 12 subjects received each of the four treatment combinations across a balanced Latin square plan. Separate error terms were used for each main effect and the two factor interaction, and consisted of the subject-by-effect source of variation. Each statistical test of main effects and interaction was therefore constructed with 1 and 11 degrees of freedom. Exact probabilities (P values) are presented for each statistical test and reflect the probability of observing the given effect size or larger effect size when there is no effect of the treatment. Standard errors reported in figures and tables around the mean value for each treatment condition represent raw single sample estimates (n = 12) and were not corrected for subject-to-subject variation. Standard errors given in the text (those associated with specific statistical tests) were based on the mean standard estimate estimated from the analysis of variance. All statistical models were constructed and evaluated in SAS (Cary, N.C.: SAS Institute, 1990).

RESULTS

Hemodynamic Responses. Responses of heart rate, stroke volume, cardiac output, peripheral vascular resistance, and mean arterial pressure across each of the four treatment conditions are presented in Table 2. Across treatment conditions, LBNP alone reduced cardiac output ($F = 13.37$, $P = 0.004$) as a result of a reduction in stroke volume ($F = 23.10$, $P = 0.0007$) despite an elevation in heart rate ($F = 6.59$, $P = 0.03$). Heart rate, stroke volume and cardiac output were similar to the baseline control condition in both G-suit inflation treatments. Compared to the control condition, mean arterial pressure was unaltered ($F = 1.39$, $P = 0.27$) by LBNP alone and increased by G-suit inflation both with and without LBNP ($F = 12.98$, $P = 0.005$). In contrast to the response of mean arterial pressure, total systemic peripheral resisitance was increased by LBNP alone ($F = 29.04$, $P = 0.0003$), but was unaltered in both G-suit inflation treatments ($F = 2.11$, $P = 0.18$).

Baroreflex responsiveness. The results for the maximum slopes of the baroreflex response function are presented in Figure 2. LBNP decreased ($F = 3.97$, $P = 0.07$) carotid-cardiac baroreflex responsiveness from a baseline of 3.8 ± 0.4 ms/mmHg to 2.7 ± 0.4 ms/mmHg without G-suit inflation while G-suit inflation, both with and without LBNP, restored the baroreflex response to 4.4 ± 0.7 and 4.3 ± 0.6 ms/mmHg, respectively ($F = 6.95$, $P = 0.02$). Figure 3 illustrates the effects of LBNP and G-suit inflation on the average carotid baroreceptor-cardiac stimulus-response relationships of all subjects.

Other baroreflex parameters. The stimulus to carotid baroreceptors was controlled and highly repeatable for all four experimental treatments (Table 1). Carotid distending pressure at the maximum slope of the baroreflex stimulus-response relationship and the heart rate-blood pressure operational point (Table 2) did not demonstrate any main effects or interactions (F 1.67, P 0.203 and F 1.248, P 0.360, respectively).

DISCUSSION

We measured carotid baroreceptor-cardiac reflex responses in 12 healthy men with and without G-suit inflation during exposure to lower body negative pressure to test the hypothesis that inhibition of vagally-mediated baroreflex control of heart rate caused by fluid distribution to the lower extremities could be reversed with application of equal counter-pressure to the lower body. Similar to previous observations (10), we found that G-suit inflation to a level equal to LBNP eliminated elevations in heart rate and peripheral vascular resistance typically induced by LBNP alone. A major finding of this study was that fluid redistribution to the lower body induced by -50 mmHg LBNP reduced the cardiac reflex response to controlled stimulation of the carotid baroreceptors by approximately 30 percent. Another important finding of this investigation was that fluid redistribution from the lower extremities toward the head induced by application of G-suit inflation with equal but opposite pressure (+50 mmHg) completely reversed the attenuation of the carotid-cardiac baroreflex response.

Our observation that fluid distribution toward the lower extremities reduced the responsiveness of the carotid-cardiac baroreflex corroborates previous findings obtained from measurement of heart rate and blood pressure responses in the supine and standing postures during phenylephrine injections (17) and during Valsalva maneuvers (15). However, a major limitation of the measurement of baroreflex response based on a ratio of the change in heart rate to change in blood pressure (i.e., HR/ MAP) with application of pharmacological and Valsalva straining techniques is that cardiovascular changes reflect an integrated response of numerous baroreflexes. Our findings of the present study extend those of previous experiments by applying a series of pulse-synchronous neck-pressure stimuli that allowed assessment of the isolated carotid-cardiac baroreflex response over most of its operational range (Fig. 3). Using this technique, we demonstrated that the capacity to buffer alterations in blood pressure over the maximal range for changing heart rate (R-R interval) was significantly reduced by acute footward fluid distribution.

It appears paradoxical that the carotid-cardiac baroreflex response would be attenuated with an orthostatic condition (LBNP) and accentuated with a condition of hypertension (G-suit inflation). However, elevations of heart rate and blood pressure during exercise are exaggerated by experimentally-induced restriction of blood flow in exercising muscles using a level of pressure similar to that applied in the present study, i.e., 50 mmHg (7). Further, isolated carotid-cardiac baroreflex responses were accentuated both at rest and during exercise when blood flow was restricted to the leg muscles by application of 50 mmHg pressure (8,9). These responses support the notion that blood flow restriction may be associated with a blood flow error sufficiently great to activate muscle chemosensors (16) and exaggerate central command input to baroreflex responses (21). An exaggerated pressor response induced by flow restriction to leg

muscles may represent a feedback control to elevate perfusion pressure high enough to reestablish adequate blood flow during conditions of ischemia. Increased responsiveness of carotid baroreceptor control of heart rate in our subjects may have reflected that a functional role of G-suit protection is to maintain an optimal baroreflex function by minimizing or eliminating the footward fluid shifts during acceleration maneuvers that typically reduce carotid-cardiac baroreflex responsiveness.

Increased heart rate and peripheral vasoconstriction during standing or other orthostatic conditions reflects elevated sympathetic activity. The carotid-cardiac baroreflex response in humans was slowed under concurrent sympathetic stimulation induced by standing and muscular exercise (20). Since redistribution of blood toward the heart by application of G-suit inflation during LBNP attenuated tachycardia and vasoconstriction induced by LBNP alone (10), restoration of the heart rate response to carotid stimulation in our subjects during G-suit inflation may reflect a reversal of sympathetic activation and its inhibitory action on carotid-cardiac baroreflex function.

PERSPECTIVES

Although a reflex-mediated increase in heart rate is rapid enough to compensate for acute hemorrhage and orthostasis, it is believed to be too slow to be effective for maintaining sufficient cardiac output during ROR of +Gz (2). With this perspective, emphasis on the contribution of G-suit pressurization has been placed on its effectiveness to mechanically increase vascular resistance and venous return. Results from the present investigation provide new evidence that G-suit pressurization may play an important role in optimizing baroreflex functions in aerial combat environments. Since carotid-cardiac baroreflex responsiveness was associated with orthostatic intolerance (4,12), including loss of vision at high +Gz acceleration (14), impairment of orthostatically-induced tachycardia caused by acute redistribution of fluid to the lower extremitites could compromise blood pressure regulation and maintenance of cerebral perfusion and lead to incapacitation of crewmembers during high-G maneuvering in military aircraft or re-entry to earth in spacecraft. The results of the present experiment underscore the therapeutic importance of G-suit application on maintaining optimal baroreflex sensitivity for blood pressure regulation.

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Table 1. Repeatability of eight neck pressure stimuli (mmHg) applied to the carotid baroreceptors during the four treatment conditions. Values are mean ± SE (N = 12).

Stimulus	Baseline	LBNP	LBNP + G-suit	G-suit
1	38.1 ± 0.3	38.6 ± 0.3	38.2 ± 0.3	38.4 ± 0.2
2	27.5 ± 0.6	27.4 ± 0.3	27.8 ± 0.4	27.1 ± 0.6
3	14.5 ± 0.5	14.6 ± 0.5	14.7 ± 0.5	14.4 ± 0.5
4	3.0 ± 0.7	2.2 ± 0.7	2.5 ± 0.9	1.9 ± 0.8
5	-8.3 ± 0.8	-9.5 ± 0.7	-9.2 ± 0.9	-9.5 ± 0.7
6	-23.1 ± 1.1	-24.1 ± 1.0	-23.4 ± 0.9	-24.1 ± 0.9
7	-41.0 ± 1.2	-41.1 ± 0.9	-41.5 ± 0.8	-41.7 ± 0.9
8	-55.8 ± 2.3	-56.8 ± 1.6	-56.3 ± 1.3	-56.4 ± 1.5

Table 2. Heart rate, stroke volume, cardiac output, mean arterial pressure, total peripheral resistance, estimated carotid distending pressure (CDP) at maximum slope, and operational point of the carotid-cardiac baroreflex under the four treatment conditions. Values are mean ± SE.

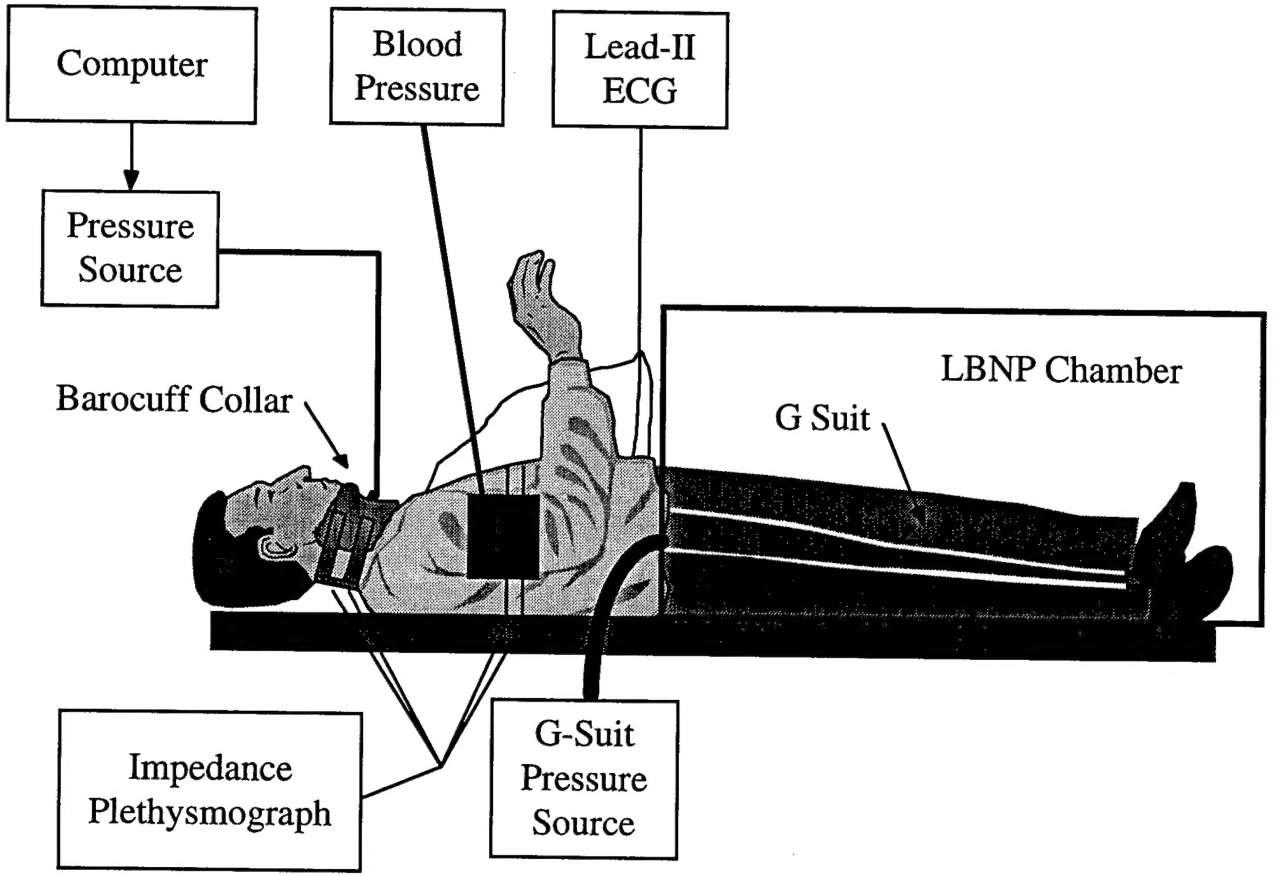
Variable	Control	LBNP	LBNP + G-Suit	G-Suit
Heart rate, bpm	64 ±2	72 ±3	66 ±3	65 ±2
Stroke volume, ml	124 ±8	73 ±6	108 ±8	122 ±8
Cardiac output, liters/min	7.9 ±.5	5.1 ±.3	7.0 ±.5	7.9 ±.5
Mean arterial pressure, mmHg	98.6 ±2.7	94.9 ±2.5	102.4 ±2.1	102.8 ±2.3
Total peripheral resistance, pru	13.3 ±0.9	19.7 ±1.3	15.9 ±1.4	13.9 ±1.0
CDP @ Maximum Slope, mmHg	135.2 ±8.7	140.4 ±7.6	138.6 ±7.3	141.3 ±6.8
Operational Point, %	38 ±8	41 ±7	35 ±5	36 ±6

Figure Legends

Figure 1. Drawing of the experimental apparatus showing a subject wearing the neck chamber and G-suit while lying in the LBNP chamber.

Figure 2. Maximum slopes (responses) of carotid baroreceptor-cardiac reflex relationship with (Yes) and without (No) LBNP in combination with (closed circles and solid line) and without (open circles and broken line) G-suit inflation. Circles represent mean values (N = 12) and lines represent raw standard errors.

Figure 3. Average carotid baroreceptor-cardiac reflex response relationships for 12 subjects during control (closed circles and solid line) compared to response relationships under experimental treatments (open circles and broken line) with LBNP (top panel), combined LBNP + G-suit inflation (middle panel), and G-suit inflation alone (bottom panel).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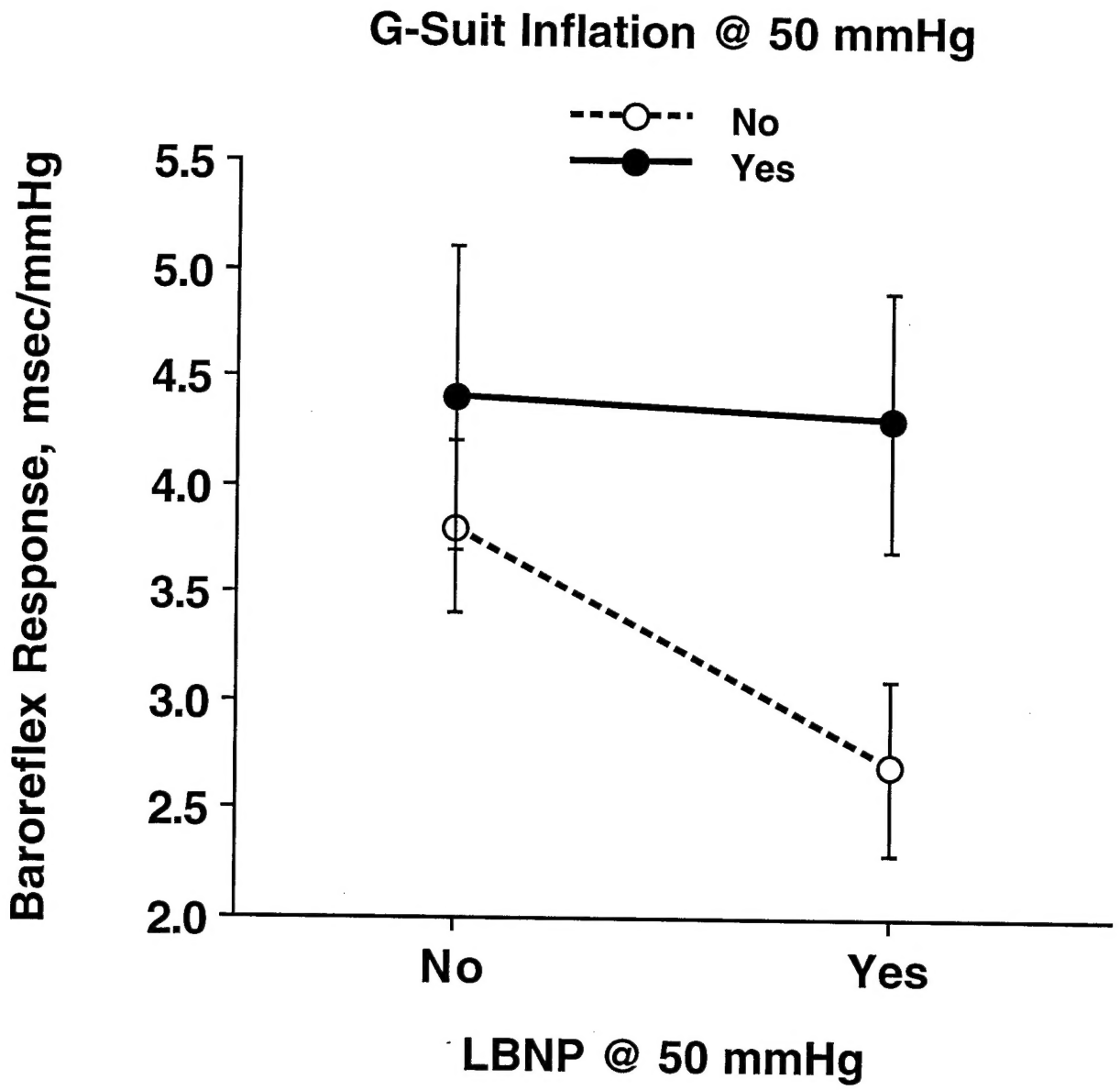
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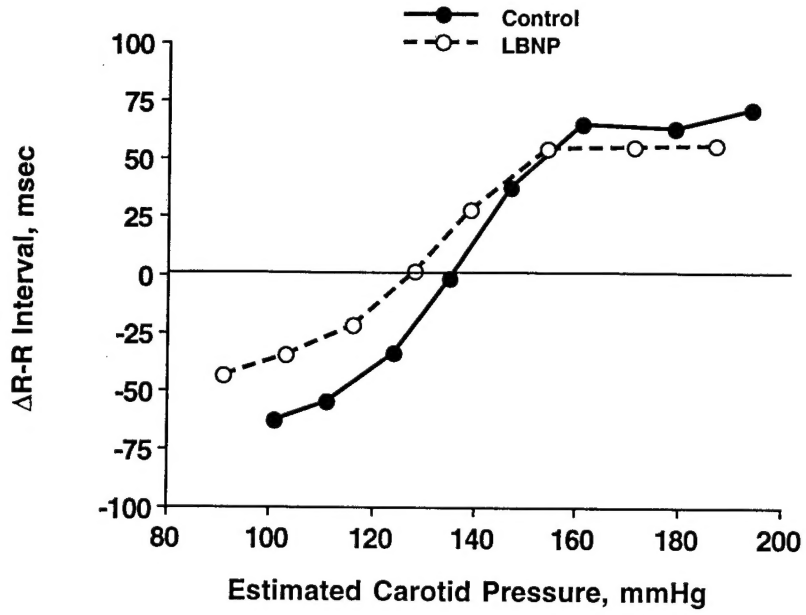
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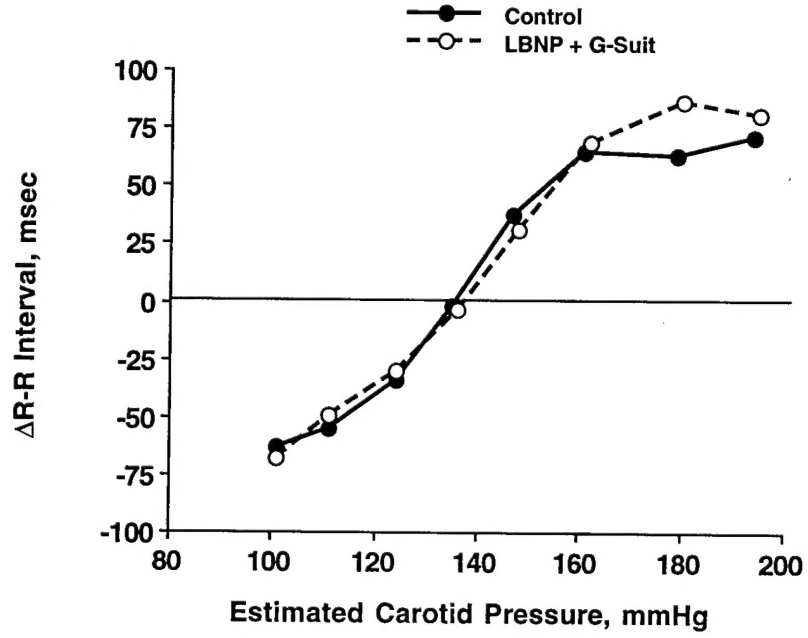
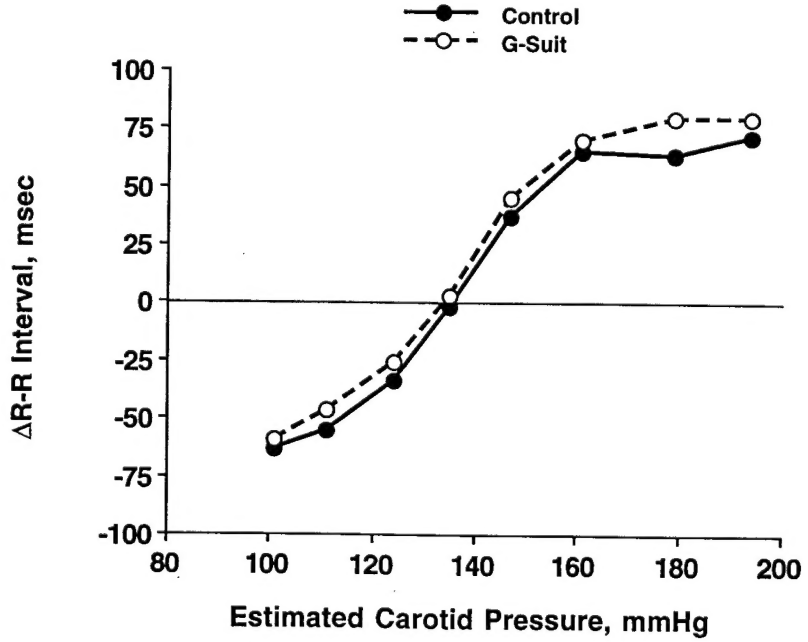
80
100
120
140
160
180
200
ΔR-R Interval, msec
Estimated Carotid Pressure, mmHg


Control
LBNP + G-Suit
100
75
50
25
0
-25
-50
-75
-100
80
100
120
140
160
180
200
ΔR-R Interval, msec
Estimated Carotid Pressure, mmHg


Control
G-Suit
100
75
50
25
0
-25
-50
-75
-100
80
100
120
140
160
180
200
ΔR-R Interval, msec
Estimated Carotid Pressure, mmHg